ARIZONA MEDICINE

Journal of ARIZONA MEDICAL ASSOCIATION

VOL. 13, NO. 3 MARCH, 1956

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Published monthly by the Arizona Medical Association. Business office at 321 Heard Building, Phoenix, Arizona. Subscription $3.00 a year, single copy 25c. Entered as second class matter March 1, 1921, at Postoffice at Phoenix, Arizona, Act of March 3, 1879.

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ARIZONA MEDICINE
Journal of Arizona Medical Association

VOL. 13, NO. 3 MARCH, 1956

Original ARTICLES

PRESENT STATUS OF TRACHEOTOMY

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Introduction:

THE performance of tracheotomies has become commonplace in recent years. In fact, one can readily name over fifty different medical and surgical conditions for which the operation is now recommended. For decades tracheotomy has been performed in patients who have shown high respiratory tract obstruction and this operation was recognized as the only satisfactory means of establishing an airway. In spite of this absolute indication, the operation has been considered dangerous and fraught with hazards and was advocated in many instances only as a lifesaving measure. When one considers that tracheotomy was usually performed in desperately ill patients and often done hastily and without proper illumination and suction, one understands why the operation was regarded in this light. There is rarely a physician, whether a general practitioner or specialist, who at one time or another in his medical career will not encounter a patient who needs this operation. The recognition of the fact that accumulation of secretions in the lower respiratory tract causes obstruction to pulmonary ventilation which leads to primary lung pathology and secondary asphyxial changes within the body constitutes the main factors responsible for the changing concepts of tracheotomy. The medical literature in recent years abounds with reports of the successful use of tracheotomy and it is to those authors[1-25] that the undersigned gives full credit and acknowledgment not only for the help rendered in the treatment of patients but also in the preparation of this manuscript.

It is the purpose of this discussion to point out the rationale for the widespread use of tracheotomy and to enumerate some of the routine procedures used in the performance of the operation, to emphasize salient complications and finally to outline the postoperative care recommended for these patients.

Indications for tracheotomy:

The indications for tracheotomy in high respiratory tract obstruction are usually well defined in the child or adult. This is particularly true if the patient is seen when the classical signs are present. Tracheotomy is usually performed in these cases without hesitation. However, if patients with acute high respiratory tract obstruction are seen relatively late, they may present signs of exhaustion and partial asphyxiation. In such patients although tracheotomy should be performed immediately, it is often unduly delayed because the signs of exhaustion and asphyxiation may be misleading.

Local pathological changes within the lungs are brought about by a patient's inability to cough secretions from the lower respiratory tract. It is estimated that the normal individual swallows between 1000 to 1200 cc. of mucus and saliva during a twenty-four hour period. In addition the cilia of the tracheobronchial tree propels secretions along the trachea until they are normally expelled to the pharynx by coughing. If these normal mechanisms are inter-

*Read before Arizona State Medical Association, Annual Meeting, May, 1955.

fered with by disease, new growth, trauma, etc., the patient will undoubtedly slowly but surely drown in his own secretions from asphyxiation. Not only may the patient aspirate saliva from the pharynx but vomitus is frequently aspirated as well. Filling of the alveolar spaces, bronchioles and bronchi with thick viscid secretions tends to interfere with pulmonary ventilation and gas exchange across the alveolar membranes. The patient's vital capacity will fall if the secretions are sufficient and if the accessory mechanisms of respiration are interfered with such as occurs in crushing injuries of the chest. Provided the patient has the strength to continue to live, other local pathological changes can occur in the lungs such as atelectasis, pneumonia, abscess, cavitation and fibrosis.

In addition to the local changes that occur within the lungs, widespread changes occur within the body and these changes will be considered now. Since the accumulation of secretions within the tracheobronchial tree and alveoli prevent exchange of oxygen across the alveolar membrane, anoxemia develops. If anoxemia develops rapidly, there is rapid loss of consciousness. A good example of this is noted when an aviator ascends too rapidly. Actually in this instance the supply of oxygen in the inspired air is diminished. When secretions accumulate within the lungs, there is a reduction in transference of available oxygen to the arterial blood. Lack of oxygen in the arterial blood, if not too great, may lead to an increased rate of breathing and increased pulmonary ventilation in an attempt for the body to compensate for this lack. However, if anoxemia is allowed to go unchecked, there is increased capillary permeability associated with irreparable damage in the central nervous system, heart muscle and other organs, and pulmonary edema.

Concomitantly with anoxemia, hypercapnia develops. The exchange of carbon dioxide across the alveolar membrane is regulated principally by the partial pressure gradients of the venous carbon dioxide and the alveolar carbon dioxide. Normally, this transference goes on rapidly and successfully because of the gradient of the two gases between the blood and the alveolar spaces. However, when secretions within the lungs interfere with this gradient, carbon dioxide is not transferred and accumulates in the arterial blood. Excess of arterial carbon dioxide is a powerful stimulator of the breathing mechanism via the respiratory centers. In contrast to oxygen lack, increased accumulation of carbon dioxide in the blood leads to headaches, restlessness, confusion, apprehension, narcosis, and to marked respiratory depression, even to failure of the respiratory centers and circulatory collapse.

As respiratory anoxemia and hypercapnia progress, respiratory acidemia also develops. When the hydrogen ion concentration of the blood tends to rise, as it does in asphyxia, pulmonary ventilation is increased in an attempt to eliminate excess carbon dioxide from the blood. Alveolar carbon dioxide tension regulates the amount of carbonic acid in the arterial blood, so if the alveolar carbon dioxide tension is shifted above 40 mm. of mercury, this increase of tension is reflected directly in the arterial blood as carbonic acid both in the free and the combined forms. This accumulation tends to shift pH of the blood towards the acid side. In order to combat this shift of blood pH, pulmonary ventilation is increased and the kidneys conserve the bicarbonate ion.

In summation, it may be stated that lower respiratory tract obstruction from secretions which cannot be coughed out by an ill patient leads first to local changes within the lungs, and to secondary widespread body changes from respiratory asphyxia. Excessive secretions within the lungs influence the exchange of oxygen and carbon dioxide across the alveolar membrances. The central nervous system cannot withstand the lack of oxygen for long periods of time without irreparable damage. This also aplies to nervous tissues in general, to the heart muscle, the kidneys, the liver, and other body organs. The body tends to reverse these changes by increasing the rate and depth of respirations. However, if this cannot be done and asphyxia continues, there is a depression of the respiratory center which in turn decreases the respiratory efforts and pulmonary ventilation, which results in further lack of oxygen, accumulation of carbon dioxide, and shift of the hydrogen ion concentration.

In addition to these effects already noted, the increased efforts of the patient to breathe against lower respiratory tract obstruction causes increased intrathoracic venous pressure which in turn is reflected as increased intracranial venous pressure. Increased intracerebral

venous pressure slows circulation which increases the carbon dioxide content of the blood, shifts the hydrogen ion concentration, and produces further lack of oxygen. These changes tend to produce increased capillary permeability, exudation of fluid, increased intracranial pressure, cerebral edema, and further depression.

Thus in the process of asphyxiation, there are many changes occurring in the body that tend to prevent the adequate supply of oxygen to the tissues. On the other hand, the body is attempting to make compensations to combat these changes and if the respiratory obstruction is relieved early, these changes are entirely reversible. If th obstruction is not relieved, all phases of respiratory compensation mechanisms will be overshadowed and asphyxiation and death will supervene. Tracheotomy should be performed early to prevent asphyxia or to combat it if it has developed. Unfortunately, there are no satisfactory laboratory tests to aid the clinician in determining exactly when tracheotomy should be done in a patient, so the clinician must rely on his experience and judgment. A safe rule to follow is to perform a tracheotomy when it can reasonably be predicted that some time during the course of the patient's illness, lower respiratory tract obstruction will develop. Tracheotomies should be performed in other patients who already have developed the inability to cough secretions out of the lower respiratory tract and who are unable to swallow their own secretions.

It is not within the realm of this paper to list all of the diseases that require tracheotomy, but rather to point out some of the diseases in which tracheotomy has been efficacious in preventing asphyxia.

Patients with bulbar or spinal poliomyelitis, or a combination of the two, may require tracheotomy. The tracheotomy should be done early in the course of the disease and frequently on a prophylactic basis. This dictum aplies to a patient whether in need of respirator care or not. If a patient with poliomyelitis shows progressing paralyses which prevent the swallowing of secretions and the coughing of secretions from the chest, then tracheotomy should be performed early. Only rarely can conservative methods be relied on for the continued mechanical removal of secretions in these progressively ill patients. Lowering of vital capacity in a patient with poliomyelitis is also an added indication for tracheotomy. The body changes from asphyxia may be the cause of death rather than the virus infection in these patients.

Comatose patients may require tracheotomy, and often this procedure may be lifesaving in itself. This statement aplies to patients who are in a coma from any cause over long periods of time. Thus patients showing barbiturate poisoning, eclampsia, tetanus, botulism, head injury, brain tumor, brain abscess, cerebrovascular accidents, and others fall in this category. The lower respiratory tract in these patients must be protected at all costs and tracheotomy appears to be the procedure of choice.

Patients with traumatic injuries about the face, jaws and neck may have sufficient upper and lower respiratory tract obstruction to require tracheotomy.

Tracheotomy has been used successfully in patients with crushing injuries of the chest to help clear the tracheobronchial tree of secretions and blood. In these patients tracheotomy further helps to alleviate pain, lessen mediastinal flutter, and reduce paradoxical motions of the thoracic walls. Performance of tracheotomy is not contraindicated even in those patients with pneumothorax, pythorax, or hemothorax.

Tracheotomy may be a lifesaving measure in patients who have undergone thyroid surgery and show the complications of recurrent laryngeal nerve paralyses or postoperative bleeding.

Patients with inhalation burns of the tracheobronchial tree may have enough edema of the mucosa to require tracheotomy; as do some patients with chemical burns in the same region.

It is a common practice in major surgical operations of the head and neck to perform tracheotomies prophylactically, not only to alleviate impending high respiratory tract obstruction, but to aid in clearing the lower respiratory tract of secretions.

Operative techniques:

Conservative methods directed at removing secretions from the pharynx and lower respiratory tract should not be used for long periods of time and should definitely not be used to the point of causing excessive trauma to the larynx, hypopharynx, trachea, and bronchi.

When possible, a patent airway should be maintained by a bronchoscope or endotracheal tube during the performance of the operation since this often prevents serious complications.

During tracheotomy some patients are able to maintain sufficient airway, while others may have to have breathing continued by mechanical means during the operation. This is done through an endotracheal tube by the anesthetist. These patients may require an extremely high percentage of oxygen insufflation during the procedure.

When possible, local anesthesia is agent of choice. However, in children and in some adults, inhalation anesthetics such as cyclopropane, ethylene, or nitrous oxygen are used.

If an emergency tracheotomy must be performed, it can often be done satisfactorily by opening the trachea directly through the neck tissues. If this is not feasible, then opening of the cricothyroid membrane should be done to save the patient's life. Later when the patient's condition permits, the tracheostomy opening should be moved to the area of the 2nd and 3rd tracheal rings.

The horizontal incision has had many advocates in recent years, although the vertical incision perhaps would serve the novice better. The isthmus of the thyroid gland as a rule must be clamped, cut, and ligated; in other instances it may be pushed either upwards or downwards. Prior to entering the trachea, all bleeding is controlled and frequently in an adult 2 cc. of 5% cocaine hydrochloride solution is injected directly into the tracheal lumen to help abolish cough reflexes. In an adult, a window is cut out of the cartilage of the 3rd tracheal ring thus creating an opening into the trachea slightly smaller than the tracheotomy tube to be inserted. In infants and children, however, a vertical incision is made in the 2nd and 3rd tracheal rings and no cartilage is removed. In the course of the operation, care is taken not to open the tissues of the neck widely so as to protect the superior mediastinum from emphysema and infection. Tracheal and bronchial secretions are removed by suction and during this maneuver coughing is produced even in an anesthetized or comatose patient. During the coughing, the wound is inspected for spontaneous hemorrhage. An appropriate sized tracheotomy tube is inserted into the trachea and the tube is held securely in place by tying cloth tapes about the neck. The wound is not sutured and may be packed lightly with gauze if desired.

Patients who require respirator care and who need a tracheotomy are intubated, removed from the respirator and the operation is performed while forced respirations are being carried out by the anesthetist. After the tracheotomy tube is inserted into the trachea, forced respirations are continued manually through the tracheotomy tube until the patient can be returned to the respirator.

Complications:

During the performance of tracheotomy, hemorrhage may be difficult to control, particularly if an emergency tracheotomy is being done. In this latter circumstance, it is better to attempt to control the hemorrhage after the tracheotomy tube has been inserted into the trachea. During tracheotomy if the patient struggles or fights for air against a closed airway and the tissues of the neck are open, the patient may develop mediastinal emphysema and uni- or bilateral spontaneous pneumothoraces. This complication can be avoided if the patient's airway is kept patent during the operative procedure. In general, the 1st tracheal ring is not disturbed during tracheotomy unless the patient is to have a largngectomy following tracheotomy, and then it is permissible to use the 1st tracheal ring as a site for tracheostomy.

In young infants and children, the pleural domes may expand into the tracheotomy wound and cover the trachea and must be pushed aside to avoid rupture of the parietal pleura.

Probably the most frequent complication following tracheotomy is subcutaneous and mediastinal emphysema. Subcutaneous emphysema can be extensive and may involve the subcutaneous tissues of the entire body. Pneumothorax may develop unilaterally or bilaterally and if it is the simple type it can be treated by aspiration of air from the pleural spaces. However, if tension pneumothorax develops, underwater drainage has to be instituted in order to re-expand the affected lung. Secondary hemorrhage in the tracheotomy wound can be serious, particularly if the blood tends to gravitate into the tracheobronchial tree. An improperly fitted tracheotomy tube may cause ulceration of the trachea even to the point of rupturing a large blood vessel.

Expulsion of the cannula from the trachea by coughing soon after completion of tracheotomy can cause death if not re-inserted into the trachea promptly. Tracheotomies jeopardizing the integrity of the cricoid and thyroid

cartilages may cause later stenosis.

When a patient is ready to be decannulized, the wound should not be primarily sutured, and if it is done ill-advisedly can lead to subcutaneous emphysema, mediastinal emphysema, and pneumothorax. This statement does not aply to fistulae that develop at the site of the tracheotomy wound after enough time has elapsed for the mediastinal tissues to be sealed off. In this instance, closure of the fistulae can be done safely and satisfactorily. In fact, some fistulae can be closed by the endoscopic removal of graulation tissue from the trachea.

Aftercare of the tracheotomy patient:

The recently tracheotomized patient should not be returned to the ward unless there are trained personnel to carry on the postoperative care. Nurses are instructed to change and cleanse the inner tracheotomy tube each hour without disturbing the outer tube. The nursing staff suctions the patient's cannula only to the depth of the tracheotomy tube and is further instructed not to suction the trachea unless an emergency arises. The doctors in charge of the patient are expected to keep the tracheobronchial tree free of secretions by suction, forced coughing, postural drainage, irrigations, etc. Moisture in the form of cold vapor is usually suplied and is supplemented by a wetting agent when indicated. The vapor and wetting agent can often be supplied by oxygen in a tent or croupette. The outer tracheal cannula is not changed until the 3rd postoperative day. Nourishment of the patient is supplied either orally or parenterally. Specific antimicrobial therapy is given. Finally, a complete set of tracheotomy instruments is kept at the patient's bedside.

Summary:

The increasing indications for tracheotomy have been pointed out. Teamwork between members of the medical and nursing staffs is essential. Indications for tracheotomy are usually clean-cut in high respiratory tract obstruction, whereas the indications for tracheotomy in lower respiratory tract obstruction are less well defined. In the performance of a tracheotomy there are greater advantages and fewer complications in the planned, orderly procedure as contrasted with the emergency operation.

Conclusion:

Early or even prophylactic tracheotomy should be recommended in selected patients who are unable to cough secretions from the lungs, in order to avoid local pathological pulmonary changes and secondary physiopathological changes within the body thereby reducing the effects of respiratory asphyxia as an added factor complicating the primary disease.

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TRUE RENAL GLYCOSURIA

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TRUE renal glycosuria is a benign condition characterized by the presence of glucose in the urine at all times, even in the fasting state, and at all levels of blood sugar. It is a rare condition. It is important to differentiate true renal glycosuria from diabetes mellitus for the treatment is entirely different. In true renal glycosuria, insulin is contraindicated and adequate carbohydrate intake must be furnished.

The criteria[1-13] for the diagnosis of true renal glycosuria are as follows: (a) A fasting blood sugar within normal limits or lower than normal; (b) glucose tolerance curve is normal or flat; (c) glucose is present in every specimen, whether voided in the fasting state or after a meal; (d) carbohydrate utilization and fat metabolism are normal.

True renal glycosuria, because it is rare and frequently confused with diabetes mellitus and with other forms of non-diabetic glycosuric meliturias, is worthy of emphasis. The following presentation illustrates a classical case of true renal glycosuria:

The patient, a 19-year-old white female, was seen in consultation because of suspected diabetes mellitus. A glycosuria was noted on admission to the hospital for an appendectomy.

The past history was non-contributory. No history suggestive of diabetes in any member of the family could be elicited.

Physical examination revealed a well developed, thin, white female who appeared moderately ill. Temperature was 100[4] orally, pulse 88, respirations 18, blood pressure 116/76, weight 108 pounds, height five feet and five inches.

Laboratory findings revealed the following: Blood count: RBC 4,00,000; WBC 16,800; differential WBC, segs 88%, lymphs 11%, eosinophils 1%; sedimentation rate 18 mm/hr. (Wintrobe); serology negative. Urinalysis: Sugar negative; albumin negative, specific gravity 1.026; microscopic examination, occasional WBC/HPF.

Blood Chemistry — Glucose Tolerance Test

Time	Blood Sugar	Glycosuria
Fasting	60 mg. %	2/-+
Half hour	90 mg. %	4/-+
1 hour	114 mg. %	4/-+
2 hour	100 mg. %	4/-+
3 hour	92 mg. %	3/-+
4 hour	80 mg. %	3/-+

The glucose tolerance curve is characteristic and typical of true renal glycosuria in that glucose was found at all times in the urine even in fasting state and at all levels of blood sugar. The glucose tolerance curve is relatively flat.

The impression of the attending surgeon was that of acute appendicitis, complicated possibly by diabetes mellitus. Consultation was requested because of the possibility of diabetes mellitus.

The patient had an appendectomy with an uneventful recovery. Her state of hydration and blood glucose level was maintained by adequate parenteral glucose and water administration. An explanation of the condition which resulted in the finding of glucose in the urine was given to the patient. It was emphasized that she did not have diabetes mellitus and needed never to take insulin. She was further cautioned that should she become pregnant, she should be under strict surveillance by her physician who should be apprised of her status of true renal glycosuria in order to avoid dehydration and acidosis.

Discussion

True renal glycosuria is also known as normoglycemic dextrosuria and orthoglycemia glycosuria.[3] It has been incorrectly termed renal diabetes and diabetes innocens.[4] Whether it is a rare or not an uncommon entity is a matter of opinion. It has been found to be a common cause of glycosuria in selectees[6-7] in World War II. There is agreement, however, that there is a high familial incidence[8-9-15] and that true renal glycosuria is not a forerunner[1-2-4] of diabetes mellitus. It is inherited as a Mendelian dominant.[15] True renal glycosuria is benign and persists throughout life once it develops.[1] The ratio of male to female patients with true renal glycosuria is three to one. It may occur at any period from infancy to old age. The pathology of true renal glycosuria is not known. No autopsy in such case has been reported. However, studies[10] of the pathological physiology in true

Presented at a Staff Meeting on August 15, 1955, at Memorial Hospital, Phoenix, Arizona.
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renal glycosuria have shown that the effective renal blood flow and glomerular filtration is normal. There is, however, a diminished tubular reabsorption of glucose in renal glycosuria which is not due to any organic kidney defect. True renal glycosuria is symptomless except during starvation and pregnancy[11] when dehydration and acidosis may develop. Such patients will exhibit glucose and ketone bodies in the urine but will have a normal concentration of sugar in the blood, (i.e.) normoglycemic glycosuria. The administration of intravenous glucose and water may be necessary to combat the dehydration and acidosis of pregnancy complicating true renal glycosuria. Patients with true renal glycosuria are usually of normal or subnormal weight in contrast to diabetic patients who are generally obese. Some[6] recommend that patients with true renal glycosuria who are underweight should have an increased amount of carbyhydrate in the diet. Other authorities[1] recommend to eat sparingly of candy, actual sugar, pastries and to avoid dietary excesses in general. If after a three-year period of observation the condition is still benign, increased laxity in regard to diet may be allowed.

The criteria[1] for the diagnosis of true renal glycosuria are as follows: (1) Glycosuria without hyperglycemia. Glucose should be found in every specimen of urine examined whether it be voided in a fasting state or after a meal. (2) Glycosuria should be independent of diet. (3) The level of blood sugar should be influenced only slightly by the ingestion of food. The glucose tolerance curve must be normal. (4) There should be no symptomatology characteristic of diabetes mellitus. (5) The type of sugar found in the urine must be dextrose. (6) There should be no progression toward diabetes mellitus. (7) Normal storage and utilization of carbohydrate as demonstrated by the respiratory quotient and metabolism before and after a carbohydrate meal. Renal glycosuria when defined in this limited sense is rare.[1] Others[3] believe that renal glycosuria is proved whenever glycosuria occurs with a normal blood sugar curve, whether the urine contains sugar at the beginning of the test or not. When defined in this sense, it is not uncommon.[3-6-7-17]

It is important not to confuse true renal glycosuria with diabetes mellitus for it may result in refusal of applications for life insurance, in needless carbohydrate restriction and futile administration of insulin with associated hypoglycemic reactions. An occasional diabetic may have a low renal threshold for glucose.[12] This is purely a coincidence. In such instance tests of the urine for sugar are not satisfactory as a guide for the treatment of diabetes. Frequent determinations of the blood sugar are then necessary. True renal glycosuria must be differentiated not only from diabetes mellitus but from other causes[13-18] of non-diabetic glycosuric melituria such as: (1) The pseudorenal glycosuria of pregnancy and nephritis, (2) alimentary glycosuria and (3) transient hyperglycemic non-diabetic glycosuria and (4) the non-glucose meliturias.

Pseudorenal glycorusia is not uncommon. It differs from true renal glycosuria in that as the blood glucose level diminished, the glycosuria diminishes and under fasting circumstances completely disappears. There is a temporary lowering of the renal threshold. Glucose may be found in the urine with blood sugars as low as 100 mg. However, when hypoglycemia is induced by insulin, glucose disappears from the urine. The following glucose tolerance test exemplifies this:

Time	Blood Sugar	Urine
Fasting	82 mg.	Neg.
Half hour	104 mg.	2+
1 hour	136 mg.	4+
2 hour	80 mg.	Neg.
3 hour	60 mg.	Neg.

The glucose tolerance curve is normal; glucose utilization is normal. Pseudorenal glycosuria is a forerunner of diabetes for there is an increased incidence of diabetes developing late in this syndrome.

In about 15% of normal pregnant women there is a lowered threshold for glucose and glycosuria appears. This condition is benign and disappears after parturition. However, it should never be taken for granted that the glycosuria of pregnancy is solely a pseudorenal glycosuria for diabetes may have its inception during pregnancy or pregnancy may occur in a woman who already has diabetes. A pregnant woman with glycosuria should have a thorough investigation to rule out diabetes mellitus and true renal glycosuria. The presence of dehydration, ketonuria, glycosuria and a normoglycosemia indicates true renal glycosuria.

Glycosuria, due to impaired tubular absorption of glucose, is observed also in patients

with nephritis and in the early phase of bichloride nephrosis.

Alimentary glycosuria (oxyhyperglycemia[3]) is the term applied to the individual who has a normal renal threshold for glucose but will have an abnormal elevation of the blood sugar after ingestion of a meal rich in carbohydrate and will excrete glucose in the urine. A few such individuals with alimentary glycosuria may eventually develop mild diabetes. Alimentary glycosuria is a condition in which the rate of intestinal absorption of glucose exceeds the body's ability to utilize glucose and the capacity to store excess glucose as glycogen. This type is common in quick emptying stomachs and after gastroenterostomy.[3]

Transient hyperglycemic non-diabetic glycosuria occurs in such "stress" conditions as acute coronary artery occlusion, cerebral hemorrhage, brain injuries.

Pathological Physiology

Glucose is filtered by the glomeruli and reabsorbed by renal tubules, so that the concentration of glucose in the glomerular filtrate is the same as that in plasma.[14] Normally none of this filtered glucose appears in the urine for the cells of the proximal convoluted tubules reabsorb all filtered glucose back into the blood stream. This reabsorption is an enzymatic reaction. However, there is a limit to the quantity of glucose that the cells can handle per minute. At a normal rate of glomerular filtration, the tubules can reabsorb about 324 mg. of glucose per minute. At certain levels of blood glucose some glucose fails to be reabsorbed and appears in the urine. This concentration is known as the renal threshold for glucose. It usually lies between 160 and 190 per 100 cc. but it varies in different persons and even in the same person from time to time. It depends on the rate of glomerular filtration. The renal threshold is not defined by the blood glucose level alone, but reflects a relationship between the total quantity of glucose filtered per unit of time and the tubular capacity to reabsorb glucose.

In a number of persons there is a defect of the tubular mechanism which is responsible for glucose appearing in the urine when the blood glucose concentration is considerably below 160 mg. per 100 cc. These persons will excrete glucose in the urine. This is non-diabetic glycosuric melituria which is defined as an excessive quantity of sugar occurring in the urine in the absence of underlying diabetes mellitus.

Studies of renal function tests in a pregnant patient[16] with true renal glycosuria revealed that, during pregnancy, fundamental changes in glucose reabsorption with changes in the plasma-glucose levels took place affecting the mechanism of reabsorption of glucose.

It has been postulated[17] that in true renal glycosuria a deficiency of tissue phosphatase may be the abnormality producing interference with the phosphorylation mechanism in the proximal convoluted tubules on which sugar reabsorption depends.

Summary

True renal glycosuria is a benign uncommon condition where glucose is found in the urine at all times even in the fasting state and at all levels of blood sugar. There is a defect in the cells of the proximal convoluted tubules resulting in a diminished capacity of these cells to reabsorb glucose from the glomerular filtrate without any other associated abnormality of excretory function of the kidneys. The recognition of true renal glycosuria is emphasized because it is frequently mistaken for diabetes mellitus and pseudorenal glycosuria. It is not a forerunner of diabetes. It requires no treatment except possibly during pregnancy. A typical case of true renal glycosuria is presented and differentiation from diabetes mellitus and other forms of non-diabetic glycosuric meliturias is discussed.

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STUTTERING

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STUTTERING is a dysrythmia of speech. It is characterized by prolongation, repetition or blockage of speech sounds, syllables or words. The flow of speech often is interrupted by a laryngeal spasm in which the vocal folds are frozen in an adducted position. Again, the interruption is due to frozen articulators or to their repetition of a sound, syllable or word without the ability to proceed to the next speech unit. Still again, the interruption is due to a failure of control of the process of respiration as required for speech. Often in such a failure the pattern of respiration reverses itself and the stutterer finds himself attempting to speak on inhalation rather than on exhalation.

In addition to these symptoms, the chronic stutterer often has developed facial grimaces and extraneous movements of head, shoulders, arms, legs and the body generally. The most severe example of such was observed in a nine-year old male. With each effort to speak, this boy raised both arms and slapped his thighs, at the same time standing on one foot and swinging the opposite one in a lateral direction. Eventually he would jump sidewise, landing on the previously swinging foot. This jump was the climax, the supreme effort to speak, which strangely enough was usually successful.

There are certain conditions under which the stutterer speaks with relative ease. He has less difficulty when talking to pets, to smaller children and when talking to himself when alone. He speaks with greater ease when reciting memorized material, when singing or when talking in unison with others. Speech comes easier for him when he feels well, when in a happy frame of mind, and the adult stutterer has little or no difficulty when mildly intoxicated.

The stutterer's speech failure increases when talking to superiors, when ill, excited or when ill at ease for any reason. Nearly every stutterer finds it difficult and frightening to talk over the telephone. Some find it more difficult to talk to strangers, while some experience a breakdown when talking to friends and even to their families.

The full-blown stutterer is beset by many psychological anamolies. He is nervous, tense and apprehensive. He is shy and socially withdrawn. He is extremely sensitive and has a general feeling of abnormalcy and inferiority – intellectually, socially and otherwise. On the physical side, he is found to be hypnotic, his reflexes are hypersensitive and bio-chemical studies have shown that ratios of certain blood chemicals fall outside the normal range. Electroencephalograms taken during stuttering spasms have revealed brain wave patterns quite similar to those of the epilectic during a seizure.

More has been written and said about stuttering than any other speech disorder, and yet there remains the widest divergence of opinion concerning its cause. Theories are numerous, as the following will show.

1. It is viewed as a neurological disorder. Some believe that due to a lack of dominance in one of the cortical hemispheres, there is an antagonism between the two in initiating stimuli to the muscles of speech. As a result, timing of the stimuli may be irregular, resulting in a "humming or hawing" of articulatory action. Or, one hemisphere may send stimuli for one kind of action while the other sends stimuli for action of another kind.

2. It is held to be the result of a conflict between the cortex and the hypothalamus. This theory holds that the inhibiting action of the cortex over the emotional center as required for speech is disrupted. There are a number of theories as to cause of such a conflict:

1. Psychoneuroses
2. Congenital instability of the hypothalamus
3. Fear of the speaking process, due to parents' amateur efforts to correct "natural" stuttering experienced by most children at some point during the early speech development
4. Unknown predisposition – organic and hereditary – with the stutter precipitated by an accident, illness or other physical or mental shock. The latter may be caused by a divorce in the family, the child's starting to school, the father's leaving for military service, unhappy home environment, etc.

3. Others believe that stuttering is caused by a bio-chemical disturbance. Blood studies of stutterers have shown endocrine imbalances involving sugar, potassium, inorganic phosphates, calcium and protein. These imbalances do not go beyond the normal range, but the ratios of the

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involved chemicals are different from those of the non-stutterer.

4. Mild hearing losses are held as a possible cause for a confusion of speech sound discrimination, resulting in a hesitation in the production of words during the speech development period. This confusion and resultant hesitation is perpetuated and fixated as stuttering.

5. Stuttering is said by some to be an oral eroticism, or a perseveration of the suckling movements of infant nursing. These movements are said to be used unconsciously in speech and are symbolic of a desire to retain the security of infancy.

6. The lack of visual, auditory or of kinesthetic imagery is held by some as the cause of stuttering. The respective advocates of these three theories believe that in order to develop and maintain normal speech one must precede the act of speech with imagery of what is to be said. Lacking such imagery, the individual never develops smoothe, anticipated expression of ideas but stumbles blindly in his efforts.

7. Some view the disorder as a poor speech habit. They believe that the stumbling, halting and repetitious speech of the infant simply becomes fixated as a habit.

These theories do not completely cover the field of speculation, but they are the most prevalent ones and will suffice to show that there is no general agreement among those in the field as to the cause of this speech malady. Many agre that in any one case any one of many conditions may cause the disorder.

Methods of correction are also numerous. They include hypnosis, psychoanalysis and psychiatry. They include strengthening visual, auditory and kinesthetic imagery. Some advocate the use of negative practice which requires voluntary stuttering, in the belief that if the individual learns to stutter at will he can cease at will. There are those who feel that correction lies in development of a dominance of one cortical hemisphere. Breath control, control of the rate of speaking, bed rest, relaxation exercies and sedation are used by others. It is quite probable that successful treatment of stuttering requires a combination of many of these methods and many agree that they do use such a combination.

Though the percentage has reached ten percent in some studies, I have found in extensive examination of school groups that approximately one percent of the elementary and secondary school population stutter. More stuttering occurs among twins and the left-handed, and male stutterers outnumber female by five to one. Practically all stuttering has its onset prior to school age and a great majority start with the beginning of speech. Most children stutter at some point during the speech development period, and this point appears to be at the time the child's ideas run ahead of his articulatory ability to put these ideas into oral speech. These children cease to stutter when, apparently, articulatory ability overtakes ideation. Some believe that those individuals who do not cease stuttering did not begin doing so because of the gap between articulation and ideation, but started because of predisposing and precipitating factors.

Despite the fact that the cause of stuttering generally has not been agreed upon, and that many methods are used, a redeeming feature is that the disorder can be corrected in most cases. My own method, which involves a number of techniques and activities and which varies as required from case to case, is successful in a great majority of cases, but there are occasional ones which do not respond to any variation of method. At best the process is an arduous and lengthy one. I have found that those six years and under require six months' therapy, while adults, strangely, and those between six and adulthood require nine to twelve months.

The chief problem facing the stutterer is the attitude that he should be left to outgrow the disorder. As a result most stutterers have become chronic before they are brought for treatment. In terms of my experience, it seems wise to give attention to the disorder if it persists, at any age, over a period of twelve months. Such persistence indicates that the individual will not make spontaneous recovery, or that if he does he will be so long in doing so that psychological involvements will occur and probably remain long after stuttering has ceased. The answer to the problem appears to be early consultation and examination and early therapy if permanency of the condition is indicated.

PHOENIX *Clinical* CLUB

The Case History in this discussion is selected from the Case Records of the Massachusetts General Hospital, and reprinted from the New England Journal of Medicine. The discussant under Differential Diagnosis is a member of the staff of the Massachusetts General Hospital. The other discussants are members of the Phoenix Clinical Club.

MASSACHUSETTS GENERAL HOSPITAL PRESENTATION OF CASE 40051

FIRST ADMISSION. A sixty-year-old retired Greek bakery worker was admitted to the hospital for repair of an inguinal hernia. He had recently felt a sudden pulling sensation in the left lower quadrant after lifting a sack of flour and noted a swelling in the inguinal region.

The past history included an operation for "locked knee" three years previously at another hospital. The patient thought there had been "a lot of blood at the time." He had been treated for syphilis after positive blood Hinton and Wassermann tests at another hospital thirteen years before admission; numerous spinal fluid examinations were negative. The serologic tests never reverted to negative, however. When he was studied for epigastric pains a year before admission, he was told he had a gastric ulcer and was treated with a diet, which he did not follow. The symptoms left, however. He had always worked in bakeries or as a laborer.

Physical examination showed a well developed and well nourished man. There were a few slightly enlarged anterior cervical lymph nodes; the chest was clear. The heart was not enlarged; there was a Grade 1, soft, apical systolic murmur. The liver edge was not palpable, but the spleen was felt 1 fingerbreadth below the left costal margin. There was a bulge in the left inguinal region that was reducible.

The temperature was 98.6°F., the pulse 70, and the respiration 18. The blood pressure was 140 systolic, 80 diastolic.

Urinalysis was normal. Routine blood studies demonstrated a hemoglobin of 16.0 gm. per 100 cc. and a white-cell count of 14,600, with 72 per cent neutrophils, 11 per cent small and 5 per cent large lymphocytes, 3 per cent monocytes, 8 per cent eosinophils and 1% basophils. There were occasional polychromatophils, and the platelets were increased. The serum protein was 6.2 gm., and the nonprotein nitrogen 32 mg. per 100 cc. The prothrombin time was 23 seconds (normal, 15 seconds), and the clotting time and clot retraction were normal. The blood Hinton test was positive. A stool was guaiac negative.

The night after herniorrhaphy, a large hematoma developed in the wound; this had to be evacuated surgically. A single 500-cc. transfusion returned the hemoglobin to its preoperative levels, and he was discharged two weeks later.

Second admission; (three years later). The patient was admitted to the hospital with the complaint of a sour stomach for many months and, because during his outpatient visit splenomegaly was again found.

He had noted a gradual decrease in appetite over the past three years, with wright loss, fatigue and occasional soaking sweats. In addition a steady, sharp, boring pain under the right costal margin anteriorly had developed about three months before admission. Also, the abdomen had gradually increased in size. He relieved this pain, which occurred an hour to an hour and a half after meals, by lying down or by taking soda and omitting the next meal.

On physical examination the liver was palpable 2 fingerbreadths below the right costal margin, and the spleen filled the left side of the abdomen. It had an easily palpable notch and was firm, smooth and moderately tender.

Urinalysis was negative. The urinary urobilinogen was 0.3 Ehrlich units per two-hour specimen. Examination of the blood revealed a hemoglobin of 11.0 gm. per 100 cc. and a white-cell count of 13,000, with 39 per cent adult neutrophils, 15 per cent band forms, 7 per cent small and 10 per cent myelocytes and 5 per cent normoblasts. There were 10 per cent reticulocytes and hypochromia, anisocytosis and poikilocytosis of the red cells and with normal pratelets. The total protein was 6.5 gm. (albumin 4.2 gm.), the nonprotein nitrogen 25 mg., the bilirubin 0.8 mg., the calcium 9.4 mg., the phosphorus 5.4 mg., the alkaline phosphatase

2.4 Bodansky units and the uric acid 5.7 mg. per 100 cc. Cephalin flocculation was three plus in forty-eight hours. A stool was guaiac negative. A quantitative blood Hinton test was positive in a titer of 1.64. An electrocardiogram was within normal limits. Roentgenograms of the chest demonstrated clear lung fields and a normal-sized heart. The bones of thorax, particularly the ribs, were denser than normal. A gastrointestinal series was normal except for displacement of the stomach by the enlarged spleen.

An iliac-crest bone-marrow aspiration was unsuccessful, but aspiration of a spinous process yielded bone marrow that showed abundant megakaryocytes, 65 per cent myeloid cells, with a shift to the right, 29 per cent erythroid cells and 6 per cent lymphocytes. No tumor cells were seen. The patient was discharged after a ten-day stay.

Third admission; (seven months later). He entered the hospital complaining of progressive abdominal swelling over the past four months, along with continued anorexia and distention after eating. He also had urinary frequency.

Physical examination was unchanged except for wasted facies, ascites, orthopnea and dullness and decreased breath sounds at the right base. There was four plus edema of the legs extending to the sacrum.

The routine laboratory studies were unchanged. The bromsulfalein test showed 5 per cent retention; the urinary urobilinogen was 3.0 units per 100 cc. and a Coombs test was negative. Other tests were as before. Paracentesis yielded 6000 cc. of opalescent, yellow fluid containing 150 monouclear cells per cubic millimeter. The symptoms were much relieved. Repeated questioning failed to reveal a history of exposure to bone-marrow toxins. Biopsy of a left axillary lymph node was reported as showing chronic lymphadenitis and reticular hyperplasia. A splenic aspiration disclosed a profusion of red-cell precursors. A barium-swallow examination demonstrated large esophageal varices. The patient was discharged to be followed in the Outer Patient Department.

Final admission (eight months later). In the intervening period he had twenty-three abdominal paracenteses at almost weekly intervals, with the removal of about 5000 cc. of clear fluid each time. Despite salt restriction, diuretics and paracenteses, fluid continued to reaccumulate, and he became weaker. About a month before admission diarrhea developed, becoming severe four days before admission. He had had non-crampy, diffuse abdominal pains and ten to twelve stools a day.

With the exception of increased emaciation, the physical findings were the same as on the previous admission.

The temperature was 98.6°F., the pulse 110, and the blood pressure 85 systolic, 60 diastolic.

Routine blood studies showed a white-cell count of 27,000, with 31 per cent neutrophils, 40 per cent band forms, 6 per cent myelocytes, 6 per cent blasts and 6 per cent basophils.

Culture of ascitic fluid grew out Staphylococcus aureus, and penicillin therapy was begun, along with streptomycin and subsequently chloramphenicol. Penicillin was given intra-abdominally. The patient gradually became weaker and less responsive over the next month, with decreasing oral intake. He was maintained with parenteral administration of fluids and antibiotics. Repeated cultures of ascitic fluid grew out Straph. aureus and nonhemolytic streptococci. He died quietly on the forty-fifth hospital day.

DR. ROGER WHITE

The case for discussion today is that of a 60-year old retired Greek bakery worker who presented with a chief complaint of inguinal hernia. During the course of the next four years he developed progressive symptoms of a disease of the hematopoietic system which led ultimately to his death.

The past history is significant only in that he had been treated for serological syphilis thirteen years previously. Three years prior to admission he had had an operation for "locked knee." He thought "there had been a lot of blood" at this time. Just what this means, I am not sure, but perhaps it has some significance in light of the hemorrhagic tendency manifested on his first admission. A year prior to his first admission he was told he had a gastric ulcer. This protocol is so long that I feel it serves our purpose best to summarize the outstanding clinical and laboratory features:

1. Splenomegaly, which was minimal at time of first admission, but reached massive proportions before death.

2. Hepatomegaly became apparent later, but as was the case with the spleen, became more and more prominent.

3. Evidence of hepatic dysfunction, as manifested by the prolonged prothrombin time on the first admission; rising serum bilirubin and urinary urobilinogen; the development of ascites and dependent edema.

4. A definite hemorrhagic tendency in the face of normal clotting time and clot retraction, elevated platelet count and an only moderately prolonged prothrombin time.

5. A moderately elevated serum uric acid level and lowered alkaline prosphatase level.

6. he only x-ray findings reported indicated a normal gastro-intestinal series at first; from esophageal varices were demonstrated at the end; and increased density of the bones of the thorax.

7. Perhaps the most significant clues are to be found in the peripheral blood:

On the first admission, hemoglobin level was normal; there was a polymorphonuclear leucocytosis. However, at this time, polychromatophilia and thrombocytosis were noted.

On the second admission, hemoglobin level had dropped to 11.0 gms.; but the smear showed 5% basophile, 10% myelocytes, 5% normoblasts. In addition, there was present a 10% reticulocytosis, hypochomia, anisocytosis, poikilocytosis and normal platelets.

8. Iliac crest-bone marrow aspiration was unsuccessful, but aspiration of a spinous process that showed abundant megaloycytes and some evidence of myeloid metaplasia.

9. Splenic aspiration disclosed a profusion of red cell precursors.

On his final admission he was in a very grave condition, after having had 23 abdominal paracenteses over the past eight months. I suspect that his complaints of diarrhea, abdominal cramps and the findings of low blood pressure can be attributed, at least in part, to sodium depletion. His blood count and peripheral smear on his last admission could certainly be interpreted as that found in myelocytic leukemia. The patient succumbed to his underlying disease with the help of peritonitis.

If I attempted to work my way through an elaborate differential diagnosis, which is warranted, it would consume ten times my allotted time. I believe that this is polycythemia vera that went through the natural history of the disease to myeloid metaplasia with a terminal picture of myelogneous leukemia and chronic osteosclerosis.

An appreciation of the protean pathological findings seen in polycythemia vera of long duration has crystallized an evolutionary pattern that encompasses all the variations seen. It has long been obvious that stimulation of a particular hematic series in the marrow often induces a reactive response in the other hematic cellular components. Localization of hemopoiesis to the marrow cavity shortly before birth results in a physiological regression of heteroplastic foci derived from the cytoplasmic reticulum. Nonetheless, these sites retain their potential functional capacity to produce differentiated hematic cells when stimulated.

In polycythemia vera the initial fundamental disturbance may very well be a reticulosis and the extra-medullary blood formation which always accompanies polychthemia vera of a sufficiently long duration is probably of autochthonous origin and is not compensatory. A concept such as this offers an attractive hypothesis for the numerous transitional clinical and pathological findings frequently encountered in polycythemis vera, leukemia, myelofibrosis, osteosclerosis and associated syndromes. Thus, the acute syndrome are usually manifested along one potential line of development, show invasive, disorganized, neoplastic tendencies of the proliferation cells and are rapidly fatal. In a few cases of acute erythremic myelosis studied, the patients always died with the characteristic marrow and blood findings of acute myeloblastic leukemia. It would appear that the erythrogenetic stimulus eventually spent itself and a myeloblastic one prevailed terminally.

In the case of erythremia the stimulus to erythropoiesis predominates, although simultaneous stimulation of the granulocytic and platelet precursor cells occur. Non-hematic increased cellular activity in the fibroblasts and osteoblasts also occur as well as reticulum cell proliferation, the latter being more obvious in the spleen. The bone marrow and peripheral blood reflect the generalized hyperactivity with panmyelosis of the marrow, and immature red and white cells and increased platelets in the peripheral blood.

The extra-osseous potential marrow space appears to undergo the same proliferative activity and the liver and spleen become enlarged due to hyperplasia of the cytoplasmic and fibrillar

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reticulum. Erythremia and myeloid metaplasia go hand in hand. The phase of polycythemia with so-called polynuclear cell leukemia or the syndrome of myelofibrosis may appear. In other case, the megakarocytes predominate with the resultant picture of acute or chronic megakaryocytic myelosis; still others display hyperactivity of the osteoblasts and bone formation encroaches upon or completely replace marrow activity, analagous to the fibrous tissue of myelofibrosis. At this phase of the disease, one may see a severe anemia, a leukemoid or leukemic blood picture, thrombocythemia or thrombopenia all in a patient who a number of years before had a severe polycythemia.

Myeloid metaplasia and myelofibrosis need not necessarily be associated with polycythemia vera, but may be idiopathic in type. Of a large series, 58% had a probable antecedent polycythemic phase, the remainder were idiopathic.

Polycythemia vera is primarily a disease of middle age. The course is one of many years' duration; a normal life expectancy may be reached. There is an asymptomatic developmental phase, the duration of which must remain conjectural although Dameshek has ascribed a period of five years for this.

Subsequently, during the stage of erythrocytosis lasting from five to twenty years with its myriad of symptoms referable primarily to hypervolemia and panmyelosis, the diagnosis is usually made.

Laboratory and clinical findings in Polycythemia Vera:

A. Laboratory
   1. Erythrocytosis
   2. Granulocytic leukocytosis
   3. Thrombocytosis
   4. RBC and WBC immaturity
   5. Panmyelosis
   6. Blood pigments normal

B. Clinical
   1. Symptoms
      a. Cardiovascular
      b. Neurological
      c. Gastro-intestinal
      d. Hemorrhagic
   2. Plethora
   3. Splenomegaly
   4. Hepatomegaly
   5. Cardiac normal; pulmonary normal

Polycythemia vera does not signify only a hypervolemic phase; however. A period of relative normalcy as far as the red cells are concerned producing the so-called remissions, have been reported. This phase should be looked upon as the first sign of diminished erthropoietic activity in the marrow and the early development of the syndrome of myeloid metaplasia. The blood picture at this time is identical to that first described in detail in this case. These red cell changes are characteristic of either polycythemia vera or the idiopathic type of myeloid metaplasia. The so-called polynuclear cell leukemia probably falls into this group of early myeloid metaplsiaa. The marrow may still be hyperplastic but instead of showing a panmyelosis, it now shows some reduction in erythroid elements with an increase in the myeloid cells, the fibrillar and cytoplasmic reticulum and the megakaryocytes. Simultaneously, with the narrow changes, the spleen enlarges and becomes the site of extramedullary blood formation. A splenic puncture at this time will reveal the findings of myeloid metaplasia.

The changes in the marrow and spleen proceed unchecked with marrow proliferation of fibroblasts and increasing encroachment upon the Erythroid tissue. Fibrosis and osteosclerosis merely signify that the stimuli, spent as far as the red calls are concerned, now show increasing activity toward the other hematic cells as well as toward the fibroblasts and osteoblasts. Red cell activity is gradually relocated to extramedullary sites. With the progression of the disease, white cell hyperactivity may predominate, resulting in a terminal picture of myeloid leukemia. In some cases the degree of megakarocytic hyperactivity may be such that the condition has been called megakaryocytic leukemia or myelosis; in others, megakaryocytic, myelocytic, fibroblastic and osteoblastic activity may all be stimulated to produce complex clinical and post-mortem findings.

I apologize for not spending more time on discussion of a differential diagnosis, but I felt that this case fitted into a neat pigeonhole, a pigeonhole which was once a cluttered maze of diagnostic nonentities such as: leuko-erythroblastic anemia, myelopathic anemia, osteosclerotic anemia, aleukemic megakaryocytic myelosis, etc.

Brinkhous and Wasserman in the Bulletin of the N.Y. Academy of Medicine, May, 1954, have written a classic entitled: Polycythemia Vera — Its Course and reatment: Relation to Myeloid

Metaplasia and Leukemia." Their concept, as discussed by me here today, brings together the multitude of variegated names and syndromes representing one common disease, erythremia.

My diagnosis:

1. Spent polycythemia Vera
2. Myeloid Metaplasia
   a. liver
   b. Spleen
   c. bone marrow
   d. lymph nodes
3. Diffuse myelofibrosis and osteosclerosis
4. Peritonitis

DIFFERENTIAL DIAGNOSIS

Dr. William H. Baker; This sixty-one-year-old man had a progressive disease involving the spleen and liver with the appearance of immature cells of both the red-cell and white-cell series in the peripheral blood and the terminal development of recurrent and relentless ascites. This process can be divided into two phases; the first occurred over a period of three years, during which the disease progressed slowly, and the second over the period of fifteen months, during which the symptoms progressed more rapidly to death.

A year before the first admission he was said to have had a gastric ulcer, which perhaps is a "red herring" because there was no evidence throughout the remainder of the history that a gastric ulcer existed. The fact that he worked in a bakery probably had little to do with the subsequent illness. The important finding was that the spleen was felt 1 fingerbreadth below the costal margin and progressively increased in size over the next four and a half years. At this time the hemoglobin was 16 gm. per 100 cc., and over the succeeding three years it dropped to a low level. Also, he had an elevated white-cell count, which is consistent with inflammatory changes within the inguinal hernia, but in view of the subsequent course I think the increased white-cell count is significant. Another pertinent point is that, in spite of the platelet increase, the prothrombin time was reported as 23 seconds with a normal of 15 seconds. It is unusual that the prothrombin time was not repeated and that vitamin K was not given before the operation. I do not know how to interpret the prolonged prothrombin time at this stage of the disease. It was suggested throughout the history that there was a bleeding tendency, and that impression is substantiated by the fact that a large hematoma developed after the operation.

In the three-year interval this patient, although he was relatively well, intermittently had weight loss, fatigue and occasional soaking sweats. These constitutional symptoms, of course, are those of any diffuse pathologic process. Tuberculosis certainly could have caused these symptoms. Malignant lymphoma, especially Hodgkin's disease, could have produced these symptoms, as could carcinomatosis. The steady, boring pain in the right costal margin that occurred intermittently through the remainder of the course is suggestive of a carcinoma of the pancreas. The fact that the pain was relieved by lying down is somewhat against a pancreatic neoplasm. Although the pain with a pancreatic neoplasm can be of any character, it usually radiates to the back and is relieved when the patient stands up or bends over.

At the time of the second admission, the liver had become palpable and the spleen filled the entire abdomen. This is quite an increase in size over three years. The patient had an anemia that was not severe. The white-cell count again was slightly elevated, and there were immature white cells and normoblasts in the peripheral blood. This is most significant, suggesting some type of disorder in the myeloid series of cells. At this time there was reticulocytosis, suggesting an increased red-cell destruction. The coombs test at a later date was negative; it was not done at that time, but the urinary urobilinogen is not a valuable test of hemolytic anemia because it is nonspecific, has to be done on a fresh specimen and is subject to a great deal of error. Moreover, it can be elevated with constipation and various other disorders that physicians are not especially looking for when they request the determination. he test is helpful but not diagnostic of any disease. I do not mean to skip over the phosphorus of 5.4 mg. per 100 cc.; that was definitely elevated and may have been related to the underlying disease. Since this determination was not repeated and confirmed it is difficult to interpret. The uric acid of 5.7 mg. per 100 cc. is at the upper limit of normal and probably consistent with the process that this man had; uric acid elevations are found in destructive lesions of the red or white cells, leukemia, polycythemia vera, myeloid metaplasia and gout.

he serologic tests were positive, and treatment was given. I am interpreting this to mean that the patient was treated for syphilis with successful suppression of activity but no reversal of the positive serologic tests.

The bones of the thorax, especially the ribs, were denser than normal. May we see the x-ray films now?

Dr. Stanley M. Wyman; The first examination of the chest shows clear lungs and a heart of normal size and shape. The ribs, however, look denser than usual; they have a uniform chalky appearance. I do not see any areas of destruction or any areas of localized increased density. Examination of the gastrointestinal tract done on the second admission shows the tremendously enlarged spleen overlying the iliac crest and displacing the stomach to the right of the midline, the duodenal cap and loop lying quite far to the right. he liver is not seen on these films. Varices are suggested at that time and are definitely present on the examination done seven months later. At the time of the second admission the chest is smaller because the ascitic fluid in the abdomen is pushing the diaphragm up. Again, the bones are definitely writer and therefore denser than normal.

Dr. Baker; Do you think you would notice that increased density if you were looking at a routine chest film? This chest film itself seems to be a little denser.

Dr. Wyman; That is a difficult question to answer now when we know the whole clinical picture; however, I think that these bones are denser than normal. This is borne out by the last film, in which the ribs are seen through the very large spleen. The increased density of the spine and pelvis is a little less striking. I do not see any evidence of a gastric or duodenal ulcer; the duodenal loop seems normal in size and shape. If there is disease in the pancreas, I do not recognize it. On the pyelogram, which is not recorded in the protocol, the right kidney is slightly smaller than usual. The calyceal system does not show any localized abnormality. The abdominal aorta is sclerotic, which is not unusual in a man of this age. There is surprisingly little degenerative change in the spine for a laborer. This later chest film again demonstrates the diffusely dense ribs.

Dr. Joseph C. Aub; The spine does not show the increased density.

Dr. Wyman; It is difficult to be sure of that. We are seeing the spine through a large, ascitic abdomen, and those bones are quite well visualized for such an examination. The ribs are denser than one expects and remind me of the picture we have seen with sickle-cell anemia with tiny bony infarcts fluorine poisoning or myeloid metaplasia.

Dr. Barker; I shall accept the fact that there was increased density of the bones.

The iliac-crest bone-marrow aspiration was unsuccessful but aspiration of the spinous process revealed abundant megakaryocytes in the bone marrow and 65 per cent myeloid cells with a shift to the right, which means an increase in maturity of the myeloid cells. The 29 per cent erythroid cells is within approximately normal limits. One has to define the type of myeloid cells seen and the percentages, before one can say that 65 per cent is absolutely normal, but certainly at this point no diagnosis of myelogenous leukemia or any other process could be made. The fact that no tumor cells were found is somewhat gratifying, but tumor cells may be present throughout the bone marrow and not be found at one aspiration.

During the final admission, the pertinent points are the extensive peripheral edema and ascites, the normal bromsulfalein test, which I consider the best single test of liver function, and the slightly elevated urine urobilinogen, which is consistent with an accelerated destruction of red cells. Paracentesis yielded 6000 cc. of opalescent fluid containing 150 mononuclear leukocytes per cubic millimeter. I assume that no tumor cells were found. I should like to know the specific gravity and the total protein of the fluid – especially the total protein in view of the fact that about 115,000 cc. of fluid was removed over the thirty-two weeks. If the total protein was high it means considerable loss of protein from the paracenteses.

Dr. Benjamin Castleman; The protein of the ascitic fluid was 2.5 gm. per 100 cc., but infection was present at that time.

Dr. Barker; This is a high level. I suspect that the serum protein fell during the last admission.

An important negative finding was a biopsy of a lymph node, which showed only chronic inflammation and slight reticular hyperplasia. Of course lymphoma could have been present elsewhere. The splenic aspiration demonstrated a profusion of red-cell precursors. I assume that

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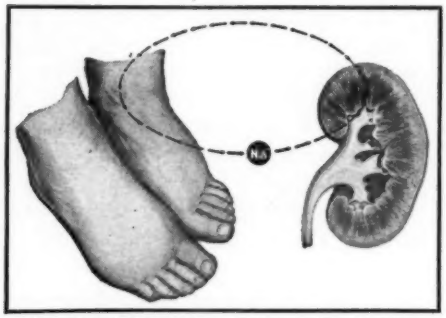

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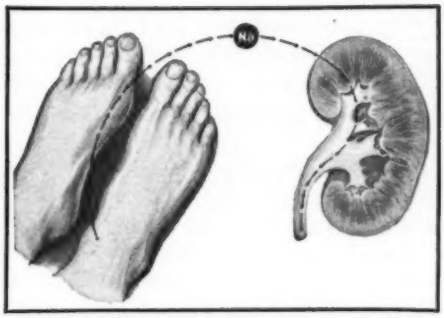

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The maintenance dosage of Mictine, as well as for initial diuresis in mild or moderate congestive heart failure, is one to four 200-mg. tablets daily in divided doses; the dosage for initial diuresis in severe congestive failure, under the conditions already described, is four to six tablets daily. For either use, it is recommended that Mictine be prescribed with meals on interrupted dosage schedules; that is, prescribing Mictine on alternate days or for three consecutive days and omitting it the next four days.

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100 per cent of the cells were erythrocyte precursors. his is significant and pertinent to this man's disease. The development of ascitic fluid, without the presence of a lowered protein or any indication of cirrhosis, and the presence of the esophageal varices suggest portal hypertension that was not of hepatic origin or due to altered serum protein but was caused either by the partial thrombosis of the portal vein or by pressue on the portal vein. I attribute the severe diarrhea to the same etiologic process – namely, venous thrombosis in the mesenteric vessels. Finally a Straph. aureus infection of the ascitic fluid developed, which I suppose Dr. Edward H. Cass, of the Boston City Hospital, would call iatrogenic. Unfortunately, as so often occurs, the organism was undoubtedly resistant to penicillin.

To put this entire progression of symptoms together, I believe that the enlargement of the spleen, which was enormous in the final stage of the disease, the gradual enlargement of the liver, the presence of immature cells in the peripheral blood and the normal bone marrow with a predominance of the myeloid series mean that this man had myeloid metaplasia. This condition has twenty synonyms or more and is at present regarded not as a disease entity but as a stage in a group of conditions called the myeloproliferative disorders. Many people believe that polycythemia vera, myeloid metaplasia, myeloid leukemia and other, more obscure myeloproliferative disorders are probably a single entity with a progression in some cases from one to the other. They certainly resemble each other in many respects, and I believe that they are probably close relations of a basic disorder. I nterpret the ascites and the eventual diarrhea as due to partial portalvein thrombosis, mesenteric-vein thrombosis and staphylococcal peritonitis. I cannot rule out pancreatic carcinoma as the "sleeper" in this clinicopathological conference, but I am not going to make that diagnosis.

Dr. Jacob Lerman; I might add that terminally the patient began to bleed from the gastrointestinal tract.

Dr. Bernard M. Jacobson; As I recall, a very obscure fever that is not mentioned in the protocol was present through most of the hospital stay. The physicians were worried about lymphoma; that was the reason for the biopsy.

Dr. Baker; Lymphoma is a better possibility in view of the fever; I did not know about that. I shall still choose myeloid metaplasia with partial portal-vein thrombosis and mesenteric-vein thrombosis.

CLINICAL DIAGNOSIS

Agnogenic myeloid metaplasia.
Peritonitis, subacute.
Toxic encephalopathy.

Dr. William H. Baker's Diagnoses

Myeloid metaplasia.
Mesenteric-vein thrombosis.
Staphylococcal peritonitis.

Anatomical Diagnoses

Agnogenic myeloid metaplasia, with extramedullary hematopoiesis in spleen, liver medullary hematopoiesis in spleen, liver, kidney and para-aortic lymph nodes, and diffuse myelofibrosis and myelosclerosis.
Splenomegaly.
Portal hypertension, etiology undetermined, with esophageal varices and ascites.
Peritonitis, acute and chronic.

Pathological Discussion

Dr. Castleman; Autopsy confirmed the diagnosis that was made both on the ward and by Dr. Baker – that is, a condition that we call myeloid metaplasia for want of a better term. The bones were all very sclerotic. A section from a vertebra showed the fat and marrow elements replaced by loose connective tissue and fibrin – what Dr. Baker called the myeloproliferative phase – and a marked increase not only in size of the bone trabeculae but also in their number, without any evidence of activity of the osteoblasts. Since there was no lining up of osteoblasts such as one expects in the conditions that are actively forming bone this much have been a slow process of bone formation, stimulated apparently by the myeloproliferative reaction in the marrow.

A physician; Were there any osteoclasts?

Dr. Castleman; Very few osteoclasts or Howship's lacunae. The bone looked inactive and perfectly normal except that there was more of it. There was little marrow left and certainly a marked atrophy of the marrow elements.

The liver weighed about 1850 gm. – only slightly above normal – and the spleen 1600 gm. – tremendously enlarged. Microscopical examination revealed throughout the spleen extensive hematopoiesis, primarily of the red cells although there were some myeloid elements.

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There was no cirrhosis of the liver. There was some hematopoiesis in the liver and some fibrosis around a few of these cells but apparently not enough to cause the portal hypertension, which I cannot explain. We found no thrombosis in any of the splenic or mesenteric veins. The ascitic fluid was voluminous and infected and was certainly a major cause of the death.

Dr. Baker; Do you think that there was pressure on the veins to account for the signs of portal hypertension.

Dr. Castlemen; That may have been one factor. We were unable to determine the etiology of the myeloid metaplasia. There was no evidence of exposure to benzene; patients whom we have seen with this intoxication show exactly the same picture — a myelofibrosis. We had always been under the impression that the hematopoietic changes in the spleen and liver were secondary and compensatory to the destruction of the bone marrow; more recently, some authorities have stated that whatever is irritating, stimulating or destroying the bone marrow elements in the spleen, liver and lymph nodes so that the reaction is a diffuse one throughout the body and not necessarily hematopoiesis secondary to bone-marrow destruction.

Dr. Wyman; Regarding the question of whether or not the bones were originally interpreted as being denser than normal, our official interpretation was as follows; "The findings are those of tremendous splenomegaly and a definite suggestion that the bones are somewhat denser than usual. The appearance suggests myeloid metaplasia."

SOUTHWESTERN SURGICAL CONGRESS

THE Southwestern Surgical Congress is meeting in Tucson April 16, 17, 18, 1956. The guest speakers will be: Dr. John Adriani, the Director of the Department of Anesthesia at Charity Hospital of Louisiana. Dr. Adriani will discuss a Symposium on Anesthesia.

Dr. Joseph Boyes, Los Angeles will discuss Hand Injuries.

Dr. Robert A. Wise will present a paper on Carcinoma of the breast.

Additional papers to cover the three day program will be presented by members of the Southwestern Surgical Congress.

AERO MEDICAL ASSOCIATION CONVENTION

THE Annual Meeting of the Aero Medical Association will be held at the Drake Hotel, Chicago, Illinois, on April 16, 17 and 18. This year's meeting will provide the most comprehensive review of current progress in Aviation Medicine ever presented. There will be 137 papers presented dealing with every aspect of the field and coming from military and civilian sources in nine countries. Of particular interest will be the section meetings which will include in their agenda panel discussions of vital topics by top authorities in each field. Those sections convening will include the following sub-specialities; Space Medicine, Civil Aviation Medicine, Aviation Physiology, Aviation Ophthalmology, Acceleration and Deceleration, Air Passenger Transportation, Noise and Vibration, Pilot Selection and Aviation Psychology, Personal Equipment, Aviation Pathology, Aviation Medical Education, and a combined symposium on Escape from High Performance Aircraft. All members of the profession are cordially invited to attend any or all of these sessions.

The Wives Wing of the Aero Medical Association, composed of over three hundred wives of Association Members will hold their annual functions on the same dates. All wives of persons in the profession are also cordially invited to attend.

MEETING NOTICE

District VIII of the American Obstetrical and Gynecological Society will hold a meeting in Tucson, Arizona on April 9th, 10th and 11th, 1956.

Postgraduate Educational Courses Five, Two-Day Regional Institutes

Postgraduate Educational Courses Five, two-day regional institution, sponsored by the California Medical Association. North Coast counties at Santa Rosa — April 5-6, 1956; San Joaquin Valley counties — at Fresno — May 10-11, 1956; Sacramento Valley counties — at Lake Tahoe, June 21-23, 1956. Address all inquiries and communications to: Dr. C. A. Broaddus, Director of Postgraduate Activities, Post Office Box A-1, Carmel, California.

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THE *President's* PAGE

PROBLEMS OF THE DOCTORS OF ARIZONA

AT the present time and the past few months, I am quite sure that all the doctors of Arizona have been disturbed by various legislative matters that have reached us through the newspapers or by other means. I know that it will be of great assurance to you, as it was to me, to know that your Legislative Committee headed by Dr. Jesse Hamer, Dr. Millard Jefferies of Phoenix, and their committee, have been on the job constantly. While it has been impossible for them to keep all the Societies informed at exactly the time that things were happening, they have done an excellent job, and I believe have carried out the wishes of the Arizona doctors.

You will be glad to know that your letters concerning House Bill 7225 have been effective in that hearings are now being held concerning this bill. However, the job is not yet completed. It is probable that constant attention must be paid to a national legislation as it relates to health.

Another subject of interest is Mental Health. Bills are being introduced in this country, and you will hear more about them.

Blue Shield is preparing, as you know, for changes which have been made in regard to including medical service in their policy. There will be certain other additions that will be discussed by your House of Delegates who constitute its (Blue Shield) corporate body, at our annual meeting.

Dr. Donald Polson of Phoenix, President of Blue Shield has done a magnificant job, and I believe that the Liason Committee of your association headed by Dr. Edward Hayden and Dr. Carlos Craig, will have many of the questions clarified that may be somewhat controversial at your annual meeting.

These then, are a few of the many problems that are being handled by very active committees. If you as an individual wish to secure information regarding the status of either legislation, or other problems, do so by either contacting your local society or your State organization. It may be that information that you desire does not reach you in sufficient time to keep you informed fully. This lag I am sure is understandable, and yet, I would invite you all to write your association, or contact it indirectly through your own representatives for any problems or information that you may desire.

Harry E. Thompson, M.D.

President, Arizona Medical Assn.

Editorial

ARIZONA MEDICINE
Journal of
ARIZONA MEDICAL ASSOCIATION, INC.

VOL. 13 MARCH, 1956 NO. 3

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The Editor sincerely solicits contributions of scientific articles for publication in ARIZONA MEDICINE. All such contributions are greatly appreciated. All will be given equal consideration.

Certain general rules must be followed, however, and the Editor therefore respectfully submits the following suggestions to authors and contributors:

1. Follow the general rules of good English, especially with regard to construction, diction, spelling, and punctuation.
2. Be guided by the general rules of medical writing as followed by the JOURNAL OF THE AMERICAN MEDICAL ASSOCIATION.
3. Be brief, even while being thorough and complete. Avoid unnecessary words. Try to limit the article to 1500 words.
4. Read and re-read the manuscript several times to correct it, especially for spelling and punctuation.
5. Submit manuscript typewritten and double-spaced.
6. Articles for publication should have been read before a controversial body, e.g., a hospital staff meeting, or a county medical society meeting.

The Editor is always ready, willing, and happy to help in any way possible.

DANGER AT THE SOURCE

THERE are 82 Medical Schools in the United States, located in only 57 cities – 82 Medical Schools for the whole 164 million of us. From them come the family doctor, all the doctors for our hospitals and communities, for industry and the armed forces, for research and many specialties. Without them – no Heart Fund, no Cancer Fund, no public health program or medical service of any kind could function. These services all depend on the doctor, and back of the doctor stands the Medical School that trained him.

There is a continuing deficit of about $8.00 per week per medical student for operating expenses in all Medical Schools, private and tax supported alike. The cost of training a doctor is now about $15,000.00 Tuition covers less than 20% of this cost. In 1955 the total enrollment in our Medical Schools was 28,583, and the operating deficit in the neighborhood of eleven million dollars.

The problem of financing Medical Education concerns every citizen of our country, but first and foremost it concerns the doctors. A minimum of $10.00 from each doctor would fulfil the obligation of the profession. This added to the amount contributed by industry to the cause each year would keep Medical Education free of subsidies by the Federal Government.

HAVE YOU DONE YOUR SHARE?

—o—

AUSTRIAN DOCTORS STRIKE

ON AUGUST 25th and 26th of last year there was an event in Austria which should be of interest to every practicing physician. On these dates the Medical and Dental professions of Austria staged a protest-strike against the present system of practice in that country. They finally told the public by open rebellion how they disapproved of a system of socialized medicine in which they now find themselves mere helpless pawns. On these two days the doctors and dentists closed their offices, saw no patients, did no surgery and signed no death certificates or other papers. Only for emergency cases was provision made. They gathered and marched by hundreds down the streets of Vienna wearing their characteristic long white coats, carrying banners to inform the public of the reasons for the strike. Handbills were passed out, public address systems and placards on every doctor's office told their story. The newspapers carried front page pictures and headlines. They asked only for a square deal and freedom to practice their profession.

To understand how they have lost this freedom we must go back to the end of the first world war. In 1918-19 Austria was not only a defeated nation but she was stripped of most of her territory. Vienna was the capital city of an empire but the empire was taken away. The people, particularly in Vienna, had no way to earn a living. To provide some medical care for this city of nearly two million people a

system of clinics and free medical service was set up. As employment slowly came back a system of socialized medicine developed and was paid for by payroll deductions. The Unions and Social Democrat party are very strong in Austria and have insisted on continuing this system.

Today 85% of the people of Austria come under this socialized medical program. Only the farmers and some of the people in the upper income brackets are not included. Now the Bureaucrats want to include these groups also. This is one of the things the doctors object to since these are the few people who still pay on a fee basis. For the 85% covered by insurance, here is what takes place. The patient takes his medical slip to a nearby doctor and receives treatment. He continues treatment for as long as three months under this same slip. He cannot change doctors during this time unless he wants to pay the new doctor. The physician is allowed to prescribe ordinary medicines, but many more expensive drugs (including penicillin) are on the chief doctor's list and before the patient can fill a prescription for one of these drugs, he must get an OK from the head doctor's office. This is such a nuisance that doctors often refer patients to a hospital for treatment where there is no limitation on the drugs used. Much of the doctor's time is taken up with forms and reports. A suburban doctor's office I visited had pigeon holes for over forty different forms.

The doctor receives his pay from the Insurance fund by sending in his collection of patients' slips at the end of each three months period. For each slip he receives 25 Austrian Schillings, the equal of about one dollar. This is his full pay for each patient seen during the three months period, regardless of how many times he has seen him. Under this system many of the doctor's patients may earn more than he does even though they do unskilled labor. But this is not the worst. A doctor cannot receive Insurance patients unless he has a contract with the Insurance Company and unless there is an area not already covered, they will not issue a contract to a new doctor. Hence there are many doctors who cannot practice and this is the freedom to practice the doctors asked for in their strike.

Obstetrics, Surgery and other hospital cases are handled in public hospitals mostly by doctors on salary. These salaries are extremely low. An assistant professor with whom I worked had been in one of the University Hospital Surgical clinics over ten years and is an excellent surgeon. He receives the equivalent of forty dollars per month. The head orderly, who comes under union wage scales,gets over twice this much. The women who scrub the floors get as much as the surgeon. So the doctors in their strike ask also for more adequate pay.

The Insurance System that covers medical practice for 85% of the people of Austria is a Bureaucracy ruled by representatives of the employers, the Social Democrat party and the Unions. The doctors are not represented. For every doctor employed there are nine non-medical employees, all with the proper political affiliations. The Bureaucrats themselves are not yet satisfied with the system. They want it to include everybody and their plan is to establish clinics and handle all medical practice through these. Doctors in these clinics, some of which are already operating, will work entirely on a salary basis. An x-ray specialist, I learned, works about five hours a day in such a clinic for about $100.00 a month. The clerks and other non-professional workers in the clinics are paid almost as much. Austria badly needs new hospitals but they are using their reserve to construct large clinics to extend this form of practice.

Payroll deductions to cover social security as well as medical care are high. In one specific instance, I was able to learn, the salary was 2160 Austrian Schillings and deductions were 440. So, the Austrian doctors' strike was a protest and warning, not against the Social Insurance System of medical care but against the impossible conditions which have developed. They are fighting for justice.

Their fight is primarily against four things:

(1) Arbitrary dismissal. Other workers have the right to a hearing if fired, but a doctor's contract can be voided at any time.
(2) Poor pay. Also other workers get extras for overtime, but not the doctors.
(3) Admission to the Insurance System of the wealthier groups.
(4) Refusal to young doctors of the right to work by not giving them contracts.

I have brought back from Austria newspapers, handbills and photographs pertaining to the protest-strike and shall deposit them in the

County Medical Society Library.

It is hardly necessary to say that we must do everything in our power to prevent medical practice in our country from falling into the hands of the bureaucrats and becoming the political racket that it now is in Austria.

Philip E. Rice, M. D.

LETTERS TO THE EDITOR

December 25, 1955
R. Lee Foster, M. D., Editor
Arizona Medicine
112 North Central Avenue
Phoenix, Arizona

Dear Doctor Foster:

On this gala day of all days, allow me to offer the finest of the season's greetings to you and your staff. Today I am enjoying a chance to catch up on my correspondence.

Many thanks for forwarding a copy of ARIZONA MEDICINE to me. You did a splendid job on my recent paper, and I appreciate your kindness greatly. While reading this issue, I was struck with the obvious and highly pleasurable attitudes expressed throughout your publication. I note that all those individuals who are concerned with this journal apparently derive a great deal of satisfaction from it. I admire this attitude greatly, and I only hope other publications would obtain a similar attitude. Then it would be a pleasure to work with them. The spirit of friendliness exudes from the pages of your splendid journal. I trust sincerely you will never lose such a wonderful spirit.

I am busily engaged in putting our forthcoming volume on RURAL PRACTICE together for our publisher, Mr. Charles C. Thomas. His son is to come here for a conference perhaps during this next week. When this book is completed and finally published, I do hope you will be able to review our volume in your excellent journal.

Many thanks for your kindness in my behalf. And with my best personal regards and all good wishes of this merry season,

Sincerely yours,
Editor, TEXTBOOK OF RURAL PRACTICE
Wallace Marshall, M. D.
Two Rivers, Wis.

Thank you, Dr. Marshall. A Happy New Year to you. — ed.

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TOPICS OF *Current Medical* INTEREST

RX., DX., AND DRS.

By Guillermo Osler, M. D.

AN Arizonan and his work have again been mentioned in 'Medical News', a very good publication of the Ciba company. Dr. Robert N. Plummer of Phoenix is quoted as having strongly underscored the dangers of delaying treatment of SPEECH DISORDERS in children. . . . The 'News' quoted his report in ARIZONA MEDICINE on the subject. They also referred to a report of ours on fungus diseases only two months ago. . . . 'Medical News' MUST be a good paper.

This year's meeting of the AMERICAN ASSOCIATION FOR THORACIC SURGERY should be stupendous. Even the announcement seems to shape that way. The Ass'n. is a super-surgical group; Dr. Paul Samson, from whom we get our data, is their king-size Secretary; and the Miami Beach hotel in which the meeting occurs is the Fontainebleau, otherwise known as the Ritz Hilton Waldorf Astoria Biltmore, the 'Most', or pretty darn large. . . . The 3-day program begins on May 7th, and includes all sorts of pulmonary, thoracic, cardiac, and cardio-vascular surgery. It should be quite an hegira. . . . (Miami Beach is in Fordila, or Foridla, or some such place in the southeast).

Two reports from the growing research center at the Los Angeles' CITY OF HOPE were given at a recent meeting of western Clinical Research groups. . . . Dr. R. T. Jordan believes that viruses inactivated by cobalt gamma radiation are more safe, stable, and potent than others. . . . Dr. Howard Bierman has found that the 'buffy coat', which can be separated from whole blood by the ADL Cohn Blood Fractionator, may act as an anti-leukemic agent It has been shown to contain an anti-hemorrhagic effect, and (in a small series of patients) it has caused clinical improvement.

It is a sad paragraph indeed which has to mention the death of DR. LOUIS J. REGAN. He spoke at the Arizona Medical Association meeting last year. . . . He was a most versatile person, being a doctor, a lawyer, a good speaker, and a fine teacher. . . . Los Angeles and Arizona will miss him in every respect.

There are medical implications in a report of the Sub-committee on Tax Policy. Mr. Greenewalt, the chairman (and president of du Pont), says that "when incomes taxes in the high brackets take so much of a businessman's dollar that he is working much harder for the government than for himself, there is considerable likelihood that he will TEND TO REDUCE HIS EFFORTS. . . . And a good thing too, we say. Less work means less tension, and less ulcers, less hypertension, etc., and a better disposition, better parenthood. . . . Even if Mr. Greenewalt can't get the Treasury department interested, he should establish a fine tie-in with the Health, Welfare and Education bureau.

Time was when the word 'prosthesis', if used at all, meant a wooden leg or a substitute arm. . . . Now it applies to such things as lucite sphere emplacements in the chest, to plastic blood-vessel grafts, and perhaps to the ordinary 'falsies'. . . . A plastic-coated glass fabric has been used by Girvin and Merendino of Seattle as a SUBSTITUTE FOR ARTERIES in animals, and recently in 3 humans. The fabric is called 'Teflon', or polytetrafluorethylene, and is said to be slick, nonwettable, of great tensile strength, and only costs $15 per yard. The most dramatic use was to replace a 17cm. length of aorta in a 77-year old man. The duration of its 'life' is still uncertain.

The Electrodyne Co., Inc., sent out a simple but surprising testimonial to a fairly recent invention. It consisted of "a partial list of users" of their Electrodyne CARDIAC PACEMAKER, or Pacemaker-Defibrillator. There were more than 200 hospitals in almost 40 states which are so equipped. . . . Most of the hospitals are on the eastern seaboard near the source of the equipment in Massachusetts. There was no Arizona hospital on the list at present.

A semi-medical book has been produced by the Biological Sciences Foundation, Ltd., called "TEA". It consists of eight papers given at a conference of the N.Y. Academy of Sciences, and they tell all about it. . . . We can remember old Dr. Loevenhart tell his pharmacology classes, "Don't expect much supper if your wife goes out to a tea party. The mild astringent effect dulls the appetite, and she will come home late and not hungry." . . . (I wish that all of pharm. had been so clearly remembered).

Another new book, from the Beacon Press of Boston, has an intriguing title, — "Standing Room Only: The Challenge of Overpopulation." . . . The author's name is fairly appropriate too, if you are a dialectician, — Sax.

Due to our neglect the 1955 CONFERENCE ON OCCUPATIONAL DISEASES was not described in this column. It is now called the 'McIntyre-Saranac Conference', with the first part of the

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name being taken from the McIntyre Research Foundation of Canada, or the McIntyre gold mines. . . . The 1955 meeting was the eighth in the past 20 years. Most of the experts in dusty diseases on this continent were present, plus Dr. G. W. H. Schepers of South Africa, now director of the Saranac Laboratory (once run by the late great Dr. Leroy Gardner). . . . Many points of interest were discussed. Probably the most controversial was the 'pre-work aluminum inhalation therapy', now in use in many mines and foundries in Canada and the U. S. Few people realize the widespread trial this method is being given, since its use has not been widely publicized. 10,000 miners have used it for 10 years in Quebec alone. The general feeling is that it has been helpful. . . . Van Ordstrand reported that 17 of 66 patients with pulmonary fibrosis had the Hamman-Rich syndrome. This makes it seem fairly common. The use of cortisone is said to be dangerous in this condition, in contrast to several other lung fibroses.

PULMONARY FIBROSIS is a topic and/or lesion about which are/could/should/must/may have more information. Several of its possible causes are enigmas, and must individually be solved. . . . Sarcoid, for instance, falls in a class of puzzles about which T. S. Eliot (an English author who is NOT a physician) could say, — "if we can never be right, it is better that we should from time to time change our way of being wrong." . . . A direct path to a better diagnosis of any fibrous lesion is lung biopsy. All that it requires is a good chest surgeon and a pathologist who does not call 50 per cent 'negative' and 50% 'nonspecific'. The hazard is now found to be very scanty, and the hazard of not making the diagnosis may include such varied items as emphysema, cor pulmonale, suppurative bronchitis, failure to use the proper therapy, the side effects of empirical steroid therapy, etc.

If you are adding a new word to your vocabulary every day (or even once a month), try the newly popular title for tranquilizers, 'ATARACTIC'. It gives a certain dignity to a relatively new group of drugs.

'XERORADIOGRAPHY' is an even newer word. It is a new type of apparatus and technic, still being perfected, and dreamed up by the Haloid Co. of Rochester, N. Y., the Battelle Memorial Institute of Columbus Ohio, and the General Electric X-ray Dep't. of Milwaukee. . . . They proclaim that it uses no water, chemical supplies, or electric power, and the roentgenogram can be fully developed in 40 seconds. . . . The procedure is a simple one, and the results transient, since a selenium-coated metal plate is electrostatically charged before exposure to x-rays. The x-rays then partially discharge areas on the plate, in inverse proportion to the density of the object. The latent electrostatic image is made visible by spraying with a powder. . . . After the plate is read, it can be wiped off and used again, eliminating storage. The advantages claimed may lead to its use in disasters and primitive areas.

The STAMINA OF CERTAIN SICK PEOPLE never ceases to be amazing, especially when the illness has seemed destructive and very grave, and when an interval of a dozen years has passed since hearing from the person. . . . We have just had a note from a man who had a terribly advanced infection of the lung with basal bronchi about the size of a man's glove. The sulfonamides once gave him some help, but the prospects were very bleak when he went back east. . . . Now he wonders about a return to the southwest, since "asthma and hay fever have been bothersome in recent years." . . . Such a person has certainly been quietly heroic in his small way.

"The substitution of oral Neohydrin for parenteral meralluride was successfully accomplished in 97 per cent of 70 ambulatory clinic out-patients with chronic congestive heart failure."*

Lawrence, W. E., Kahn, S. S., and Riser, A. B.: South. M. J. 47:105, 1954.

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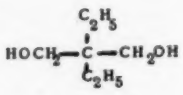
ARIZONA *Pharmaceutical* PAGE

WHAT'S IN A NAME

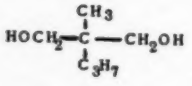
by Joseph A. Zapotocky, Ph. D.
Pharmacy College, University of Arizona

SHOULD a drug be more to you than just a label with which you may associate certain actions or indications for use? Does the chemical name of the drug have some meaning for you and does it enable you to classify it chemically and therapeutically? Can you compare chemically related drugs? Do you critically evaluate a drug with a new name and decide from its composition whether it is an innovation, a modification of something already on the market, or merely a new garb for an old drug?

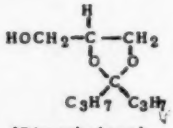
Rarely does the average physician have time to make these distinctions. He is usually not sufficiently versed in chemical terminology to know and recognize the chemical name of the drugs he prescribes. Many manufacturers may introduce the same drugs under different titles. For example, although Mephenesin is the official name for 3-o-toloxy-1,2-propanediol, it may appear on the market as Tolserol, Dioloxol, Lissephen, Tolulexin, Oranixon, Sinan, and other titles. If you were to receive literature on a new drug called "Nodreem" which turned out to be 3-o-toloxy-1,2-propanediol, would you recognize it? If you received literature on a drug "Nopane" which was aminopyrine disguised as 1-phenyl-2,3-dimethyl-4-dimethylamino-5-pyrazalone, would you recognize it and know that although it is an effective analgesic it had the well known disadvantages of the coal tar analgesics?



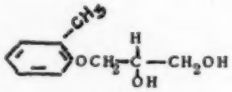
(Prenderol)



(Miltown, Equanil) the dicarbamate of the above structure.



(Dimethylane)



(Mephenesin, Tolserol, etc.)
Tolseram is the b

More recently a drug appeared on the market under the names of Miltown and Equanil. Both are brands of the dicarbamate of 2-methyl-2-n-propyl-1,3-propanediol. Although it is a new drug, were you able to associate it immediately with Mephenesin? Did you notice its very close similarity to Prenderol, 2,2-diethyl-1,3-propanediol? A very close observer would have been able to include Dimethylene in the same class.

Once the relationship of the structure of these compounds is noted, then the reasons for their similarity of action begins to appear.

The majority of pharmacists do very little compounding today. But because of a longer period of education and experience they have become consultants on the thousands of drugs which they are called on to dispense. Their comprehensive and specialized knowledge of drugs can help unravel the complexities of drug terminology for the physician. This ability to recognize and classify the thousands of names already in the prescription room and the hundreds added yearly is an indispensable trait of a good pharmacist.

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REFERENCE: 1. Silcox, L. E., *A.M.A. Arch. Otolaryng.* 60:431, Oct. 1954.

Notes from the EDITORS PEN

ARIZONA-AMEF — CONTRIBUTION

Council of your Association at a meeting held in Phoenix, November 27, 1955, directed that an amount equal to $5.00 per active member as of January 1, 1955, be contributed by the Association out of its general fund to the American Medical Education Foundation, 50% thereof to be allocated to medical schools comprising the Western Regional Compact for Higher Education. This amounts to $3,525.00.

The officers and directors of the American Medical Education Foundation have expressed their sincere appreciation for the generous contribution the Arizona Medical Association has made to relieve the present crisis facing the medical schools. Arizona has again demonstrated its leadership.

Schools receiving an equal share of the grant comprising the Western Regional Compact include the University of Washington Medical School, University of Utah College of Medicine, University of Oregon Medical School, University of Colorado School of Medicine, University of California at San Francisco, University of California at Los Angeles, Stanford University School of Medicine, University of Southern California School of Medicine and College of Medical Evangelists School of Medicine.

It was further stated that the other medical schools of the United States will be sincerely appreciative of their share of this generous contribution.

ELECTION ALERT — 1956

1956 is an ELECTION year that could have a great impact on the medical profession in the years ahead. You and your friends have a grave responsibility: be qualified to vote, know the issues, know the candidates' position, make up one's mind for him to vote, know when and where to vote — and VOTE!

In Arizona, Primary election is currently scheduled for Tuesday, September 11th (Arizona will elect a Senator — last day to file July 23rd); general election Tuesday, November 6th. Check up on your registration NOW.

H.R. 7225 SOCIAL SECURITY

This might be an effective approach to expressing medicine's views clearly and with brevity. Doctor George C. Lincoln, of Woodstock, Connecticut, concerned with all the implications of the proposed amendments embodied in H.R. 7225 of which you have all hear much, reached for his prescription pad and under his signature prescribed the following:

"Rx One vote against Bill H.R. 7225

"Sig: Take as often as necessary to prevent economic indigestion and other dire effects should the Bill be passed.

"Prescription may be refilled and also given to friends."

The "prescription pad" could be a most unique courier, similarly used, to impress OUR Congressmen of medicine's views on important legislation, such as H.R. 7225. Let's try it on our Senators. It may make Arizona healthier for them.

HILL-BURTON GRANTS

The Department of Health, Education and Welfare reports that as of December 31, 1955, Hill-Burton grants completed and in operation in Arizona include 15 projects at a total cost of $12,050,437, (federal contribution included $3,650,050) supplying 884 additional beds. Under construction is one project of a total cost of $102,739 (including federal contribution of $51,369). Approved but not yet under construction are five projects at a total cost of $2,018,960 (including federal contribution of $771,500) designed to supply 30 additional beds. In December 1955, two projects were approved. St. Joseph's Nurse Training School, Phoenix, total cost (estimated) $1,075,960 (approved federal share $300,000); and John C. Lincoln Hospital, Phoenix, estimated total cost $140,000 (approved federal share $70,000) designed to supply ten additional beds.

ANNUAL MEETING

Remember the date — April 25 through 28, 1956; the place — Chandler, Arizona — San Marcos Hotel; and a GOOD TIME to be had. Make your ROOM RESERVATIONS now and count upon being present.

AMERICAN CANCER SOCIETY

The Arizona Division of the American Cancer Society has just completed its Fourth Annual Cancer Seminar in Phoenix at the Paradise Inn. For the second year we commend this organization on the presentation of this excellent and instructive program. The faculty that has been made available to the local men has been superior. It was surprising to see a number of them return in this capacity. Their presentations were stimulating but a great variety of view-

points might prove helpful. An unusual aspect of the session was the consideration given to the mental status of the cancer patient and the doctor.

We would like to congratulate the Arizona Division of the American Cancer Society and Drs. Bregman and Barger in particular for their efforts in establishing these postgraduate courses and encourage them to continue their program in the coming years.

The registration at the past meeting was approximately 350 doctors. The attendance included many more when one considers Technicians and Nurses. However, of the 800 doctors in our state there were less than 200 registrations from Arizona. This program is worthy of the consideration of all the doctors of Arizona in the future.

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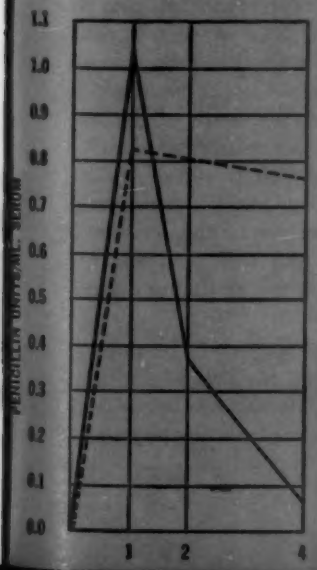

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Organization PAGE

CIVICS

Norman Ross, M.D.

WE'VE BEEN PLEDGED

(Arizona Medicine Organization Page, November, 1955)

The Governor's Arizona Mental Health Research Committee met with its Advisory Committee and the Committee of the University and Colleges for Mental Health Research for the State of Arizona, on January 28, 1956. Following the welcoming address by Governor Ernest W. McFarland, Dr. C. H. Hardin Branch, Head of the Department of Psychiatry, University of Utah Medical School, and Project Administrator for WICHIE (Western Interstate Commission for Higher Education), the designated Western Regional Survey Agency for the Council of State Governments to conduct an area survey of the Mental Health Training, Research, and Prevention, was introduced by D. W. Melick, M.D., Secretary of the Arizona State Medical Association and Secretary of the Arizona Division of the Western Interstate Commission for Higher Education.

Dr. Branch's address is an excellent explanation of these regional mental health surveys and mimeographed copies of same can be obtained on request from the Governor's Arizona Mental Health Research Committee, 620 Professional Building, Phoenix, Arizona.

When we consider that Public Law No. 182, which is a part of an intensified attack on Mental Health, was passed by the Congress of the United States without a dissenting vote, and that mental illness is termed the number one health problem by the American Medical Association Council on Mental Health, we should study both Dr. Branch's explanation and this law.

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ARIZONA TUBERCULOSIS AND HEALTH ASSOCIATION, INC., 111 East Willetta Street, Phoenix, Arizona, Helen E. Watkins, Executive Director.

The annual meetings of the Arizona Tuberculosis and Health Association and the Arizona Trudeau Society will be held April 20-21, 1956 at the Hotel Adams, Phoenix.

J. Arthur Myers, M.D., Minneapolis, Minnesota, an outstanding authority on tuberculosis in children will be the banquet speaker Friday evening, April 20. He will participate in the programs the following day.

Julius L. Wilson, M.D., Director of Henry Phipps Institute, Philadelphia, and Director – American Trudeau Society – Medical Education Program – will appear at both the general session and the scientific session on Saturday afternoon, April 21.

The general sessions are open to the physicians and public. The scientific sessions, though related to tuberculosis detection, treatment and control, will present subject matter of interest to all physicians. An invitation to attend is extended to Arizona Medical Association members by the Arizona Tuberculosis and Health Association and the Trudeau Society.

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MARICOPA COUNTY SOCIETY FOR CRIPPLED CHILDREN AND ADULTS, INC., 7211 North Seventh Street, Phoenix, Arizona (Easter Seal Society) LeRoy Larson, Clinical Director.

The Samuel Gompers Memorial Clinic, owned and operated by the Maricopa County Society for Crippled Children and Adults, Inc., has in cooperation with the office of Vocational Rehabilitation recently constructed an audiology center at the clinic. This center will bring to the valley the finest in the way of sound-treated rooms and equipment for the most discriminate hearing testing required and will be ready to give necessary service to the state in the very near future. Referrals for hearing evaluations can be made to the audiology center by any qualified physician and any qualified agency may feel free to avail themselves of the services of this center after preliminary testing indicates or warrants the need for further and more discriminate testing procedures.

All testing and examinations will be done on a fee basis – based upon the cost of operating the audiology center. Referrals can be made directly or through an agency contracting or using the services for their clients.

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Convention Headquarters

ARIZONA MEDICAL ASSOCIATION

65TH ANNUAL MEETING
April 25, 26, 27, 28, 1956

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President

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Book REVIEWS

PHYSICIAN'S FEDERAL INCOME TAX GUIDE, 1956 Edition by Campbell & Liberman Published by Channel Press, Great Neck, New York, Price $2.50.

THE MANY physicians who are familiar with the earlier editions of this fine book will be glad to know that the 1956 edition has just been published. Those who are not familiar with the previous edition should know that this is a book designed to aid you in assembling and evaluating the necessary information to fill out your income tax return and to aid you also in actually filling in the various sections of this rather complicated return. The various types of taxable income are treated individually and we note that over forty different types of possible non-taxable income are listed which should be of some help in keeping the physician from needlessly paying taxes on income which is not actually taxable. Various deductions are treated individually and in the arrangement of the text of this book, each paragraph dealing with a particular subject is headed by a statement printed in italics which you can readily evaluate as to pertaining to your situation or not in order that you may know whether or not it is necessary for you to read that particular paragraph. For instance one paragraph is entitled "If you incurred gambling losses during 1955, read this:". Another, "If you purchased bonds at a premium, read this:". And another, "If you receive no income from annuities or pensions in 1955 you may now skip to section 2 of this chapter:". The text is liberally illustrated with sections from a specimen report. On the outside cover is a printed guarantee that you will save sixty times the $2.50 purchase price or your money will be refunded or if you do not chop hours off your tax return time, your money will be refunded. After looking through this well arranged booklet, I do not believe that there are going to be many takers for this guaranteed refund.

R. L. F.

HAWAIIAN CENTENARY

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Woman's AUXILIARY

COME TO THE CONVENTION

ON behalf of the Committee for the Twenty-sixth Annual Convention of the Woman's Auxiliary to the Arizona Medical Association, we wish to take this opportunity to urge all members to attend the meetings to be held in Chandler, at the San Marcos Hotel April 26th, 27th, 28th, 1956.

Maricopa County Woman's Auxiliary President, Mrs. George A. Williamson, and the Maricopa County members will be hosts for the guests from other counties.

State Auxiliary President, Mrs. Roy Hewitt, and her Committees have arranged a particularly interesting social and informative program.

State Convention Committee Chairmen are: Mrs. Charles L. von Pohle, Chairman, Mrs. William E. Ragsdale, Jr., Co-Chairman, Mrs. Donald Buffmire, Reservations, Mrs. Seth Douthett, Registration, Mrs. M. E. Stern, Finance, Mrs. Clifford Goodman, Transportation, Mrs. L. A. Stapley, Jr., Publicity, Mrs. Robert Delph and Mrs. Robert Erickson, Decorations, Mrs. Lloyd Kent, Courtesy.

The following tentative program has been announced by the Program Committee. A printed program will be available at the Convention.

Friday, April 27th—10:00 A.M. Formal opening of the General Session of the Auxiliary, in the Paris Lounge, San Marcos Hotel—Mrs. Roy Hewitt, President, presiding; followed at 12:30 P.M. by a luncheon in the main dining room, honoring Mrs. Mason G. Lawson of Little Rock, Arkansas, The National President of the Woman's Auxiliary to the American Medical Association, who will be the speaker.

Saturday, April 28th—10:30 A.M. Brunch and Fashion Show by SOLEDAD of Phoenix, Scottsdale and Chandler, at the Arizona Country Club, in Phoenix, followed by a short business session and installation of 1956-57 officers. The day's festivities will wind up with the State Presidents Dinner-Dance given at the San Marcos Hotel by the Doctors.

A cordial invitation is extended to wives of all doctors whether or not they are members of the Auxiliary, to attend this State Convention.

INVITATION TO CONVENTION

The 26th annual convention of The Woman's Auxiliary to the Arizona Medical Association will be held at The San Marcos Hotel, in Chandler, April 26th, 27th, 28th.

Maricopa County is the hostess Auxiliary, headed by Mrs. Charles L. von Pohle, chairman and Mrs. William E. Ragsdale, Jr., co-chairman. They and their committee have planned many social events and business sessions too.

We will be honored by having our National President, Mrs. Mason G. Lawson, of Little Rock, Arkansas, as our guest.

As your president I extend a cordial invitation to all doctor's wives in the State to attend our convention.

Irene Hewitt (Mrs. W. R.)
President

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Chiles, George H.	AL 4-1211	618
Hensing, C. R.	AL 3-6603	819
Johnson, William J.	AL 3-1866	919
Lee, Joseph A.	AL 4-2345	517
Leibold, John P. (Child only)	AL 8-3142	711
Miller, Eugene H. E.	AL 3-4312	702
Pafford, Ernest, Jr.	AL 3-3807	718
Pafford, Ernest M.	AL 3-3807	718
Scott, E. M.	AL 8-4277	918
Sitkin, Frank L.	AL 4-2345	517
Smith, Franklin R.	AL 4-3257	717
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Steninger, Milo C.	AL 4-3561	621
Voorhees, Joseph P.	AL 8-3320	802
Oral Surgery		
Bairo, Louis P.	AL 3-9362	711
Borah, Charles E.	AL 3-7414	814
Peterson, Ralph G.	AL 2-6313	719
Stallcup, L. B.	AL 4-3675	418
Orthodontists		
Jensen, Albert J.	AL 3-2327	617
Moffat, Richard P.	AL 4-1322	1017
Periodontists		
Burke, Wm. G.	AL 8-4644	818
Creamer, R. Dean	AL 3-6534	820
McGuire, Vaughn S.	AL 3-6718	1118
Physicians & Surgeons (M.D.)		
Cardio-Respiratory Diseases		
Randolph, Howell	AL 4-3146	1005
Dermatology		
Mackoff, Sam M. (A).	AL 2-0379	920
Medigovich, D. V.	AL 3-6617	905
Endocrinology		
Raddin, Joseph B.	AL 2-3577	619
Eye, Ear, Nose & Throat		
Barnet, E. G.	AL 4-3341	1120
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Gaskins, Duke R.	AL 3-8582	922
Johnson, James L.	AL 4-2841	806
Melton, B. L.	AL 3-8209	605
McCurdy, Gordon J.	AL 3-8209	605
Nelson, W. J.	AL 3-5121	1011
Reese, Forrest L.	AL 4-3341	1120
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Barker, Robert D.	AL 3-2176	710
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Dysart, Palmer	AL 4-8483	1108
Felch, Harry J. (Ob.S.)	AL 3-1151	705
Hussong, R. W. (Ob.G.)	AL 3-2883	520
Matanovich, M. (I.S.U.)	AL 3-6509	422
McKenna, J. F. (A).	AL 4-2174	910
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Deitchman, Morris (Ca.)	AL 2-1802	1008
Gatterdam, E. A. (A).	AL 4-2174	910
Hamer, J. D. (Ca).	AL 4-2174	910
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Harbridge, D. F.	AL 3-5604	822
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Meyer, Wallace M.	AL 2-2822	903
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Barrett, Robert S.	AL 3-1491	606
Kow iski, L. J	AL 4-2850	906
McGrath, Wm. Bede.	AL 3-5559	811
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Foster, R. Lee	AL 3-4105	507
Kennedy, John W.	AL 3-4105	507
Watkins, W. W.	AL 3-4105	507
Thoracic Surgery		
Melick, Dermont W.	AL 4-3146	1005
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VNB Car-Park	AL 3-2835	
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